

STAGES OF SHOCK

SHOCK : A profound disturbance of circulation and metabolism, which leads to inadequate perfusion of all organs which are needed to maintain life.

COMPENSATED NONPROGRESSIVE SHOCK 30 sec -48 hrs

A. Decrease in BP leads to an increase in the sympathetic responses

1. Skin vasoconstriction
2. Vasoconstriction to the kidneys ↓ Urine Output → Renin (Angio I, II
→ Vasoconstricts)
(Aldosterone → Na reabsorption and H₂O retention)

3. Release of Epinephrine and NE

4. Increases Heart Rate and the force of the contraction

B. Release of Aldosterone to reabsorb Na⁺ and H₂O follows Na⁺

C. Release of ADH to reabsorb H₂O

D. Hypoxia leads to an increase of blood flow to the tissue but has a harmful effect by increasing blood flow

DECOMPENSATED PROGRESSIVE SHOCK

Loss of 15-20% of blood volume and the deterioration of the cardiovascular system

A. Decrease of BP below 60 mmHg leads to myocardial ischemia and a weakened heart muscle and a decreased cardiac output and a further decrease of BP setting up a positive feedback loop

B. Decrease of BP below 50 mmHg leads to general vasodilatation causing further loss of BP

C. Increased Hypoxia leads to the increased permeability of the capillaries due to loss of hydrostatic pressure causing the loss of blood plasma into the tissue decreasing blood volume.

D. Intravascular Clotting -Decrease in blood volume leads to a ↓ in the velocity of the blood and an ↑ viscosity. This allows the platelets to aggregate in the vessel leading to clot formation, causing obstructions (↑ Viscosity = ↓ Velocity)

E. Cellular Destruction is caused by the lysosomal rupture and ↓ in the activity of the mitochondria, active transport and general metabolism.

F. Build up of lactic acid lead to acidosis with pH dropping to 7.35- 6.80 or lower

IRREVERSIBLE SHOCK

Heart deteriorates until it can no longer pump and death occurs.

Signs of shock

- 1 Vasoconstriction of the vessels of the skin cause cool clammy and pale skin
2. Increase in epinephrine leads to tachycardia and sweating
3. Reduced cardiac output and vasodilatation leads to weak rapid thready pulse

4. Cerebral ischemia alters mental status
5. Reduced urine output due to increase of aldosterone and ADH leading to increased thirst
6. Acidosis due to builds up of lactic acid
7. Nausea due to vasoconstriction of the digestive system – shunting of blood flow to Vital organs.

I. Cardiogenic Shock (Pump Shock)

Caused by any type of heart failure

CHF or MI
 Infections of the myocardium or pericardial sac
 Drug toxicity

Clinical manifestation:

Mental impairment
 Elevated systemic and pulmonary preloads
 Tachycardia
 Low BP and cardiac output with no loss of fluid
 Elevated preload
 Dusky skin color –delayed capillary refill
 Decreased Cardiac Output and Cardiac Index
 Note – only shock with ↑ systemic vascular resistance

Treatment:

Mortality rate is high
 Treat the underlying cause
 Angioplasty and thrombolytic therapy have improved chances
 Because the heart is weak and there is no loss of fluid watch IVs, any fluid
 Positive inotropes – Dobutrex
 Afterload reducers – Nipride, Nitrates

volume overload would put more work on the heart further damaging heart muscle

II. Prime Shock – Hemorrhagic Hypovolemic

Hemorrhagic – Internal vs External

Hypovolemic shock : most common

Caused by the loss of body fluids

Direct
 Loss of whole blood – Hemorrhage

Loss of plasma – Burns
Indirect
Loss of Interstitial fluid – Diaphoresis, diabetes, emesis or diuresis

Clinical manifestations:

Poor skin turgor
Thirst
Oliguria
Low systemic and pulmonary preloads
Tachycardia

Treatment:

Fluid replacement

III. Pipe Shock: Neurogenic, Septic, Anaphylactic, Maldistribution of Blood Flow

Neurogenic Shock or Spinal Shock

Pooling of blood within the peripheral vessels with no loss of fluid

Caused by

Spinal cord injury
Spinal anesthesia
Drugs which inhibit sympathetic nervous system thus inhibiting peripheral vasoconstriction

Clinical manifestation:

Flaccid paralysis
Absence of reflexes
Hypotension
Bradycardia
Paralytic ileus
Loss the temperature regulation

Treatment:

Treat the underlying cause
May last days to months
Vasoconstrictors (Dopamine)
Terminates with the return of reflexes, hyperreflexia and spasticity

IV. Septic Shock

Cause: Invasion of a microorganism mainly gram negative but some maybe gram positive, as in the case of Toxic shock, also maybe the result of a fungi or a virus.

Microorganism release of toxin inflammatory response

Remember the key to inflammation is vasodilatation which allows fluid into the interstitial spaces out of the blood vessel. Also during an inflammatory response the body's temperature and metabolic rate increase.

Maldistribution:

Some areas of the body will be hyperperfused, (warm shock) while other areas will be underperfused

Massive peripheral vasodilatation- pink warm flushed skin

Increased intravascular fluid shift to the third spaces leads to increase in blood viscosity (increased Hematocrit)

Microemboli formation leads to decrease tissue perfusion

Signs and Symptoms:

Increased temperature due to pyrogenics

Increase WBC's

Increased metabolic rate

Increased Heart rate (bounding pulse)

Pulmonary edema

Decreased Urine output

Metabolic acidosis from the build up of lactic acid

V. Anaphylactic Shock

Caused by exposure to an antigen/allergen leading to acute and life threatening hypersensitivity reactions.

Antigen →release of vasoactive mediators (histamine, heparin, cytokines)

→massive vasodilation, peripheral pooling and ↑ capillary permeability

Clinical Manifestations

Anxiety

Dyspnea (D/T Bronchoconstriction)

GI Cramps

Edema

Uticaria

Pruritis

Hypotension (D/T massive vasodialation)

Treatment

Priority : Maintain Airway

Benadryl (antihistamine) – oral or IV depending on severity and patients level of Consciousness

Epinepherine – IM or IV

(sympathomemetic →vasoconstriction and bronchodilation)

